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14. Saul Rosenthall to O. M. Salate, colleague of Herman Schwan, August 8, 1968.

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RADIO-FREQUENCY AND ELF ELECTROMAGNETIC ENERGIES

A HANDBOOK FOR HEALTH PROFESSIONALS

R. Timothy Hitchcock · Robert M. Patterson

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To Leslie, Benjamin, Michael, and especially Sandra

To Meredyth, Bradley, and Lauren ... and to Pat

Radio-Frequency and ELF Electromagnetic Energies
A Handbook for Health Professionals

[redacted]

[redacted] extremely low frequency (ELF) [redacted] and uses electric power.

Tailored specially for the working health professional, *Radio-Frequency and ELF Electromagnetic Energies* is a practical guide to understanding, evaluating, and controlling the human health effects of radio frequency (RF) and extremely low frequency (ELF) electromagnetic fields. You'll find here all you need to know about radiation safety, from the basic physics on how to set up a safety program. Discover how these electromagnetic fields interact with matter, the reported biological impacts, significant sources of and efficiency and extremely low frequency fields, and the equipment needed for safety evaluation.

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The perfect blend of applied information and theory, this handbook is essential reading for industrial hygienists who work in [redacted] physicists, health officers, researchers, and electrical engineers.

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CONTENTS

Chapter

3

❑

BIOLOGICAL EFFECTS OF RADIO-FREQUENCY FIELDS

3.1 GENERAL

❑ Radio-frequency (RF) electromagnetic energies may affect a number of systems, organs, and functions, as compiled in Table 3-1. These biologic effects have been the topic of a number of worthwhile reviews over the last few years (Michaelson 1971, 1974, 1980, 1982a, 1982b, 1986, 1991; Sliney and Conover 1975; Cleary 1977; Lin 1979; Heynick and Polson 1983; Michaelson and Lin 1987; Osepchuk 1983; Petersen 1983; Elder and Cahill 1984; Curtis and Nichols 1983; ILO 1985; Polk and Postow 1986; NCRP 1986, 1988; Heynick 1987; Beers 1989; Elder et al. 1989; Gandhi 1991; Saunders, Sienkiewicz, and Kowalczuk 1991a, 1991b; Wilkening 1991; WHO 1993). Because there have been so many recent reviews, this chapter does not give a comprehensive treatment of this topic but places emphasis on long-term studies, certain nervous system effects, reproductive effects, and cancer in test animals, while epidemiology, clinical studies, and incidents will be our focus for human beings.

The studies selected for inclusion in this review fall into the following categories. (1) Studies or avenues of research important in the establishment of the present human-exposure criteria, such as effects on animal behavior. (2) Studies that may assist the health professional in answering inquiries from concerned individuals, such as questions dealing with reproductive and developmental effects. (3) Studies that illustrate specific points of interest or controversy, or demonstrate the complex nature of an avenue of research, such as in vitro studies of calcium efflux.

While reviewing this information, it is important to keep in perspective that a biologic effect does not necessarily equate to an effect that is hazardous to health. For example, exposure of test animals with microwaves (MW) may cause concentrations of certain biochemicals to fluctuate within normal homeostatic limits but without any functional impairment in normal processes. Also, the biologic significance of an effect may not be understood well enough to establish it as potentially hazardous. Although this does not

Table 3-1. Systems Involved in RF-Induced Biologic Effects

Special senses
Central nervous
Neuroendocrine
Reproductive and developmental
Hematopoietic
Immunologic

rule out the possibility of the effect ultimately being found adverse, conversely it does not provide proof that the effect is detrimental.

3.2 NONTHERMAL EFFECTS

As discussed in the preceding chapter, the recognized mechanisms of interaction produce thermal effects following absorption of RF energy by ionic, molecular, and/or cellular structures. This energy is dissipated as heat that increases body temperature. Simply, the body's thermophysiologic response includes increased blood flow, vasodilation, and an increased sweat rate. Some bioeffects that are thermal include neuroendocrine effects, teratogenic effects, testicular effects, and cataract formation.

According to Schwan (1992), questions about thermal versus nonthermal effects were raised in the 1930s during the development of short-wave diathermy. Since that time, there have been few answers in the area of nonthermal effects, and there has been an ongoing controversy dealing with the existence of interaction mechanisms that produce athermal or nonthermal effects. Athermal effects have been defined by Elder (1987) as physiologic changes in which the core body temperature is not elevated, but a quantity of energy is absorbed sufficient to activate thermoregulatory receptors and cause a physiologic response. Athermal effects include immune and endocrine effects stimulated by exposure to low-intensity RF.

Nonthermal effects are responses due to low levels of exposure that cause no significant thermal input and, hence, no significant change in body temperature (Elder 1987). Suggested nonthermal effects include electroencephalographic changes, effects on cellular membrane potentials, field-force effects, and calcium flux in brain tissues.

Electric-field-force effects include pearl-chain formation, orientation of asymmetric particles, movement of particles in inhomogeneous fields, and deformation, destruction, fusion, and rotation of cells (Teixeira-Pinto et al. 1960; Saito and Schwan 1961; Schwan 1971, 1982, 1987; Michaelson 1985). These effects have been observed in in vitro experiments where the E field interacts with polar or nonpolar structures and produces an orienting effect. Pearl-chain effects have been observed in a number of cells including leukocytes, erythrocytes, and bacteria (Johnson and Guy 1972; Schwan 1982). Simply, the pearl-chain phenomenon refers to the alignment of cells or particles with one another in the direction of the imposed E field (Presman 1970). Other postulated nonthermal effects include millimeter wave effects and membrane interactions (Schwan 1971; Barnes and Hu 1977; Taylor 1981; Michaelson 1985; Motzkin 1987).

In 1986, the National Research Council convened a select panel of scientists, who were not involved in the debate concerning thermal and nonthermal effects, to review that topic. The panel concluded the following:

> Bioelectromagnetics research has produced abundant reports of a wide variety of individual biological responses to low-level nonionizing radiation. At least three mechanistic biophysical theories have been offered to explain how non-thermal interactions could develop. However, the connections among the various experimental findings and the theoretical constructs do not yet lead to a comprehensive conceptual structure for the reported phenomena sufficient to enable an evaluation of the significance of the theories (National Research Council 1986).

3.3 ANIMAL STUDIES

To be included here, studies had to report important exposure parameters, such as the SAR, frequency, exposure duration, and radiation intensity. In some cases SAR was not reported, but sufficient information was available to allow its estimation. Estimates were made using various editions of the *Radiofrequency Radiation Dosimetry Handbook* (Johnson et al. 1976; Durney et al. 1978; Durney, Massoudi, and Iskander 1986). In some cases, SAR estimates made by the EPA in *Biological Effects of Radiofrequency Radiation* were utilized (Elder and Cahill 1984).

Results from animal studies form the basis of the human-exposure standards presently in use in the United States and throughout much of the world. Animals that have been used in studies include rats, mice, guinea pigs, cats, dogs, rabbits, hamsters, monkeys, birds, and arthropods, with rodents being the species most frequently used. Although there are many good reasons to use rodents (relatively inexpensive, easy to handle, and thorough biologic characterization), they have a number of anatomical, physiological, metabolic, nutritional, and behavioral dissimilarities when compared with humans (Oser 1981). It is possible that these differences may limit the utility of the experimental data or complicate the interpretation of the data. An anatomic difference that may have some bearing on RF absorption in rodents is that they are fur bearing. More important, rodents do not sweat, a major thermophysiological cooling mechanism in human beings. Obviously, this is a point because the experiments are trying to model an RF-induced thermal response in human beings by extrapolation from the rodent.

Most studies were short term and evaluated acute effects at a single frequency and a single SAR. There have been relatively few long-term experiments. Due to the complexity and expense of these experiments, they have been funded largely by the military and a few government agencies. Although this has produced some high-quality studies, it has had the effect of limiting the scope of the experiments in regard to potential exposures received by many workers. As would be expected, studies funded by the military examine specific frequencies associated with military applications. Some studies, like those of radar workers, may have wider application than others. Quite a few studies have been performed at 2450 MHz, and although a number of short-term studies have been done at 27.12 MHz, no long-term experiments have been reported at that frequency, which is an important frequency in both industry and the public sector. Few long-term experiments have been performed that model exposure at frequencies within the human whole-body resonance part of the spectrum. This becomes important in terms of the human exposure standards, which are based largely on the results of short-term experiments at relatively few frequencies.

RF biologic effects studies are complex undertakings. In addition to the retinue of highly trained specialists who are required to formulate, implement, and evaluate animal studies, RF studies require the involvement of individuals with specialized electrical or radiation engineering expertise. Hence, much of the data that have been generated in the areas of dosimetry and bioeffects have come from electrical or bioengineering departments at major universities or from specialized research arms of the federal government.

The studies also have some hardware requirements that are unique to studies with physical agents. Figure 3-1 shows a shielded, RF-anechoic chamber that is used to expose a single animal. It is equipped to study effects of short-term MW exposures on behavioral end points and contains a styrofoam restraining chair to hold monkeys. Styrofoam is used because it has a low relative permittivity and will exert a minimal influence on the RF fields.

Obviously, materials that influence or perturb the field cannot be used because they affect the outcome of the experiment by modifying the RF dosage by disturbing (perturbing) the field. For example, cage design, materials of construction, and spacing between cages are important considerations in RF bioeffects research. Typically, cages are made from materials that have nonperturbing characteristics, such as plastics or glass. However,

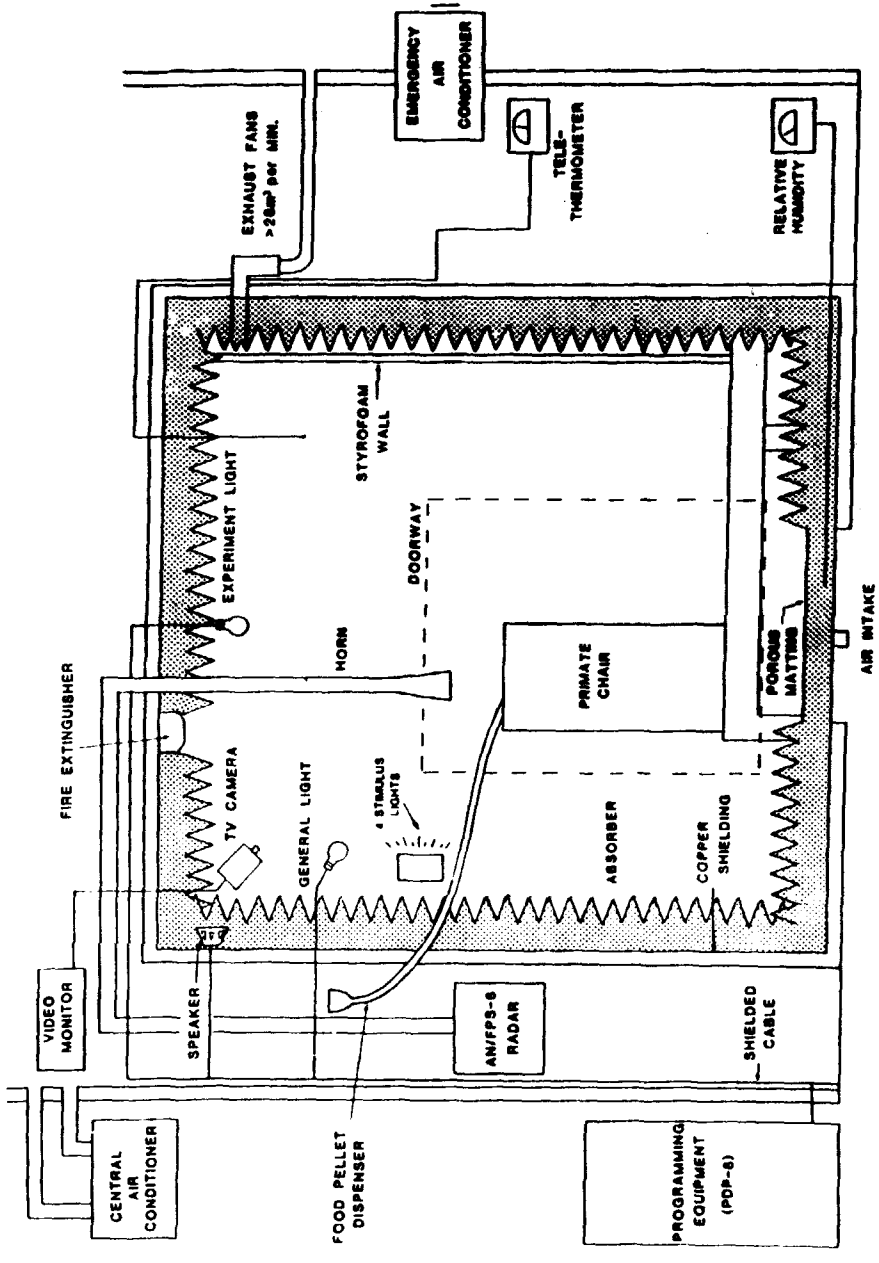

Figure 3-1. Radio-frequency semi-anechoic chamber used in behavioral experiments with primates. (From D'Andrea, Cobb, and de Lorge 1989. Used with permission.)

plastics may still perturb the field (Lin, Bassen, and Wu 1977), although when an animal is introduced into the cage, the overall SAR may not be greatly changed. Ho (1978) explained "that while the Plexiglas holder alone can set up a large standing wave, the presence of the animal, which is highly lossy dielectric material, reduces the influence of the Plexiglass holder." Material thickness must be controlled, because for a given material it has been shown that perturbations increase with thickness (Lin, Bassen, Wu 1977; Ho 1978).

Spacing between conductive objects, like cages or animal bodies, may be important because of possible reflections. This has led to criticism of studies that use so-called gang exposure techniques, because the dosimetry is usually defined for one animal, and many are exposed in close proximity. However, Berman et al. (1985) found no difference in the LD_{50}/24 h in test mice whether they were exposed singly or in groups of 16. Nevertheless, most modern research attempts to minimize mutual coupling between animals. This is achieved by building a radiation chamber for each animal (Guy et al. 1980) or by locating the cages at an optimum spacing distance that will minimize intercage scattering (Bonasera, Toler, and Popovic 1988).

The size of the cage may affect the experiment, as evidenced by a study of the potential for MW radiation to act as a tumor promoter. Four groups of animals were used: high- and low-MW dose, and two nonexposed groups. Of the control groups, one was a sham-exposed group, while the other was treated similarly but housed in smaller cages. This "chronic confinement stress" group consistently developed cancer more quickly than the low MW-dose group but not as rapidly as the high-dose group (Szmigielgski et al. 1982). Although the vehicle was a small cage, the mechanism probably involved a generalized stress reaction due to neuroendocrine compromise.

Special provisions must be made for water and food provided during the experiment, so that containers do not influence the field. The reasons for taking these precautions are well illustrated by a multigenerational experiment performed at 60 Hz. The researchers observed that the exposed animals had significant reductions in body weight; however, they suggested that food and water were introduced in a fashion that may have produced electric shocks to the animals (Marino, Becker, and Ullrich 1976). It was noted by Bridges and Preache (1981) that these "results could have been greatly influenced by the presence of this spark discharge." When the experiment was performed in a manner that controlled the potential for spark discharge, the influence on body weight was not observed (Marino et al. 1980). To avoid potential problems, some researchers have withheld food and water from both exposed and control groups during the irradiation period, although this may produce a weight reduction in the animals (Lary, Conover, and Johnson 1983). Other researchers have located water and food in the corner of the cage farthest from the radiation source to minimize perturbations (Bonasera, Toler, and Popovic 1988).

In some cases, the animals may require anesthesia or restraint so that a specific anatomic region may receive local exposure. It is possible that the use of anesthesia or restraint may modify the RF dosage or the measured response of the animal. For example, workers for NIOSH found that restraint and use of a rectal temperature probe elevated the measured temperature in the control animals 0.5°C during a half-hour period (Lary et al. 1982). Lai et al. (1987) have reviewed combined effects of RF with some anesthetics, while Williams et al. (1984) observed that anesthetics used in some experiments designed to evaluate permeability changes of the blood-brain barrier may have been an interference.

The general laboratory environment may also have an effect on the outcome of an experiment. For example, EPA researchers found that when the ambient temperature was raised from 20 to 30°C, the estimated power density necessary to achieve the LD_{50}/24 h was cut in half (Berman et al. 1985).

In studies of developmental effects, it was found that handling techniques affected maternal weight gain (Nawrot, McRee, and Staples 1981) and fetal weights (Nawrot, McRee, and Galvin 1985). To minimize po-

tential complications due to handling, researchers include an adaptation period in the studies, to allow the animals to become familiar with their surroundings, handling, and the test procedure (behavioral studies). In studies of bloodborne end points, animals may be cannulated, then allowed to adapt. Cannulation allows blood collection during the study without performing stressful invasive techniques at each collection period. To minimize potential stress-induced effects due to handling prior to sample collection, researchers may collect blood samples after test animals experience a brief adaptation period in a sampling box (Toler et al. 1988).

3.3.1 Long-Term Studies

The results of long-term studies are compiled in Table 3-2. One of the earliest studies, by Prausnitz and Susskind (1962), found no differences in body weights of mice, but the controls had a higher death rate than the exposed animals. The authors speculate that this could be due to the MW-induced thermal stress (average 3.3°C rectal temperature rise during exposure), essentially a fever, which may have helped the exposed animals combat a pneumonia outbreak that occurred during the experiment. Exposed animals exhibited a greater incidence of testicular degeneration and of leukosis and leukemia.

Spalding, Freyman, and Holland (1971) exposed mice inside a waveguide where the E field was perpendicular to the long dimension of the waveguide. Except for white-blood cell (WBC) count, bloodborne parameters in the control group showed a decreasing trend. In a comparison of paired differences between the groups, voluntary activity and bloodborne parameters were not significantly different. The exposed mice lived an average of 19 days longer than the control mice, although this was not statistically significant.

Rats were exposed to electromagnetic pulses (EMP) where PRF = 5 Hz, rise time was 5 ns, and fall time = 550 ns; 250 million pulses were delivered during the experiment, with the peak E-field strength equal to 447 kV/m; 320 male and 20 female rats were exposed, with an identical number serving as controls. There were no significant differences in bloodborne end points, tumor incidence, and type and macroscopic and microscopic evaluations during necropsy (Baum et al. 1976).

Dumanskij and Sandala (1974) studied the effects of 50-MHz UHF radiation (0.0006–10 $\mu W/cm^2$) on white rats and rabbits, and 2.5-GHz SHF radiation on rabbits (0.5–10 $\mu W/cm^2$). Radar exposure was simulated at 10 GHz (width = 1 μs, PRF = 20 and 1000 Hz) and 1, 5, and 10 $\mu W/cm^2$. Baseline measures were determined prior to exposure of the UHF group, which had no controls. Results showed blood cholesterol and sulfhydryl levels decreased throughout the experiment, while 17-ketosteroid in the urine increased. This latter effect, an increase in 17-ketosteroid levels, could possibly be due to recovery of the animals from the stress of a new environment since, apparently, animals were not allowed to adapt before exposure.

Researchers at the University of Utah were unable to replicate the findings of Dumanskij and Sandala (D'Andrea et al. 1979, 1986a, 1986b). Adult, Long-Evans rats were allowed 4 weeks to adapt to experimental conditions, then were randomly divided into exposed and controls. Significant increases were found in total sulfhydryls at weeks 6 and 10. In the study by Dumanskij and Sandala, sulfhydryl levels decreased. D'Andrea and colleagues (1979) conclude that "there is no satisfactory explanation for these discrepant findings or for the functional significance of this parameter for the animal." Significant differences were found in red blood cells and white blood cells for the week 6 sampling period, but not for weeks 2, 10, or 14, which was attributed to sampling error or was an artifact.

In another study, D'Andrea et al. (1986a) found no significant differences in body mass, food and water consumption, blood levels of cholinesterase, and poststudy evaluations for electrolyte levels, ion gaps, and CO_2. A difference was observed in the levels of 17-ketosteroid between exposed and control groups during the adaptation period, which may be

Table 3-2. Long-Term Studies

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d × min)	Effects	Reference(s)
Mice (male)	9200 Pulsed	50[a]	100	295 × 4.5	Testicular degeneration and leukosis or leukemia in exposed mice; higher death rate in controls	Prausnitz and Susskind 1962
Mice (female)	800	1.3[a,b]	43	175 × 120	Exposed mice lived average of 19 d longer	Spalding, Freyman, and Holland 1971
Rats	EMP	NR	See text	658 × 1380	No differences	Baum et al. 1976
Rats and rabbits	50 2500 and 10,000	NR	See text	120 × 600 or 120 × 720 120 × 480	Decreased sulfhydryl levels and cholinesterase, increased 17-ketosteroid levels	Dumanskij and Sandala 1974
Rats (male)	2450 CW	1.23 ± 0.25	5	80 × 480	Significant difference in activity	D'Andrea et al. 1979
Rats (male)	2450 CW	0.14	0.5	90 × 420	Differences in 2/4 behavioral measures	D'Andrea et al. 1986a
Rats (male)	2450 CW	0.7[c]	2.5	90 × 420	No differences in physiologic end points; significant differences in behavioral end points	D'Andrea et al. 1986b
Rabbits (sex not specified)	2450 CW	1.2 to 2.2	10 ± 3	40 × 480 to 85 × 480	Changes in RBC count and dietary habits	Ferri and Hagan 1977
Rabbits	2450 CW	1.5[d,e] 17 (head)	7 10	180 × 161	Reduced eosinophils and WBC count	Guy et al. 1980
Rats (male)	2400 CW	2[e]	5	90 × 60	No differences in hematologic measures and organs	Djordjevic, Lazarevic, and Djokovic 1977

Table 3-2. (Continued)

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm²)	Exposure (d×min)	Effects	Reference(s)
Rats (male pups)	100 CW	2.96 to 2.55[d]	46	22×240 42×240 97×240	No differences in most measures; significant transient differences observed	Smialowicz et al. 1981
Rats (male)	2450 Pulsed	0.15 to 0.4[e]	0.48	750×1260 (25 mo.) 390×1260	Elevated adrenal mass; differences in immune competence; no differences in numerous end points	Guy et al. 1985
Rats (male)	2450 CW	0.15 to 0.4[c,e]	0.48	180×1260 360×1260	Change in progenitor blood cells	Chou et al. 1985
Rats (male)	435 Pulsed	0.3 to 0.35[c,e]	1	180×1320	Dopamine levels reduced; other hematologic parameters not different	Tolen et al. 1988
Dogs (female)	24,000 Pulsed	NR	24	440×400 360×990	Reduced weight gain; differences in cholesterol	Deichmann et al. 1963
Mice (female)	2450 CW	2 or 6.8	3 or 10	≥ 572×60	High SAR group had significantly shorter life span	Liddle, Putnam, and Huey 1994

[a]Estimated based on Durney, Massoudi, and Iskander 1986.
[b]An estimated WBA-SAR = 1.3 W/kg would occur if the magnetic-field vector is parallel with the animal's length, although this estimate may be somewhat inaccurate due to the standing-wave environment within the waveguide. This estimate derives some support from the finding that $< \frac{1}{2}$ W was absorbed by the 12 mice. Assuming 20 g/mouse suggests a SAR of about 2 W/kg.
[c]Whole-body average (WBA).
[d]For the pups, SARs decreased with increasing body mass, from around 3 W/kg between post-partum days 1-10, to around 2.5 W/kg between days 31 to 50. SARs were estimated at around 2 W/kg for the dams.
[e]WBA-SARs varied with animal mass as the animals matured.
CW, continuous wave; EMP, electromagnetic pulse; NR, not reported.

due to stress induced by handling and housing conditions. Significant differences were observed in two of four behavioral tests, but these may be of "little biological significance" (see Section 3.3.4.6). In a third report, D'Andrea et al. (1986b) found statistically significant differences in behavioral tests, which will be reviewed in more detail later.

FDA researchers restrained New Zealand white rabbits and exposed them at 2450 MHz, finding that exposed animals had a significant reduction in red blood cell (RBC) counts, food intake, but not water consumption. There were no significant differences in WBC count, ocular changes, and coat condition. The authors conclude "that chronic low-level exposure of rabbits to MW irradiation can cause peripheral RBC changes and affect the dietary habits of the animals" (Ferri and Hagan 1976). However, this observation cannot be generalized because of the small size of the test group.

Guy et al. (1980) also exposed New Zealand white rabbits at 2450 MHz. Statistically significant reductions were found in albumin, calcium, and eosinophils in the exposed rabbits. No significant differences were noted in the other 38 parameters evaluated. Guy notes that three significant outcomes out of 41 measured end points is near that expected by chance at the 0.05 level. At thirty days post exposure, no significant changes were found in bloodborne end points; however, albumin levels in the exposed animals had decreased, and globulin levels had increased. Pathologic findings showed a significant difference in the myeloid/erythroid ratio in bone marrow samples from the sternum: "The biologic importance of this finding is questionable since the hematologic (erythrocyte and leukocyte counts) parameters did not differ between treated and control rabbits" (Guy et al. 1980; McRee et al. 1980). These results are not generalizable because of the small number of animals used in the study.

Djordjevic, Lazarevic, and Djokovic (1977) exposed rats using a gang exposure technique that introduces some uncertainty into the SAR estimate. There were no differences observed in body weight, rectal temperature, hematocrit, mean cell volume, hemoglobin, erythrocytes, leukocytes, neutrophils, lymphocytes, and organ (spleen, liver, heart, brain, and testicles) histology.

EPA researchers exposed 20 gravid Sprague-Dawley rats from day 6 of pregnancy to parturition for 4 h/d in a transverse electromagnetic (TEM) transmission line. There were no significant differences in body weight, although RF-treated pups were inclined to be larger than sham-exposed animals. There were no differences in RBC and WBC counts, hemoglobin and hematocrit, immunology, mutagenesis (dominant-lethal test on sperm), and neurologic development. Significant differences were observed in eye opening, weight of the medulla oblongata, and regional concentration of acetylcholinesterase (Smialowicz et al. 1981). The effects on brain weight and the enzyme appear to be transient in nature, and they may represent normal biologic variation.

A lifetime study that evaluated an extensive number of end points (155 parameters) was performed at the University of Washington for the U.S. Air Force (Guy et al. 1983, 1985; Guy, Chou, and Neuhaus 1983; Chou, Guy, and Johnson 1983; Johnson et al. 1983, 1984; Kunz et al. 1983, 1984, 1985; Chou et al. 1992). The study was designed to simulate, in male rats, chronic exposure of humans at 450 MHz and 1 mW/cm^2 (Guy et al. 1983). One hundred Sprague-Dawley rats were exposed with an equal number as controls. Pulsed MW radiation (width = 10 μs, PRF = 800 Hz) was square-wave modulated at 8Hz.

In 14 open-field assessments of behavior, there were no differences between the groups, except for a reduction in activity of the exposed animals during the first session (Guy, Chou, and Neuhaus 1983). Plasma corticosterone (a stress indicator) levels were not reliably affected. Levels were elevated during the first of five sampling periods in the exposed animals, while levels in the controls were elevated during the third. At 13 months, MW-exposed rats exhibited a stimulatory effect on the immune system as indicated by statistically significant increases in splenic B and T cells. There were no differences in T and B cells at 25 months. Results at 13 months

also indicated a significant difference in stimulation of spleen lymphocytes to select mitogens (substances that stimulate lymphocytes to proliferate). No data were available at 25 months due to failure of the lymphocyte cultures to grow (Kunz et al. 1983). Hematologic parameters were not reliably affected. There were no differences in serum chemistry, thyroxine, body weight, food, water and oxygen consumption, and CO_2 production. Organ mass was not different for animals sacrificed at 13 months, but it was significantly elevated for the adrenal gland for 10 exposed animals at 25 months. The added mass was associated with adrenal tumors. This finding is reviewed in Section 3.3.9.1.

In a follow-up investigation, 2 groups of 20 male rats each were exposed as shown in Table 3-2 (Guy et al. 1985). The results did not replicate those of the earlier study for plasma corticosterone, increases in splenic T and B cells, and stimulation of spleen lymphocytes to specific mitogens. An effect found in both 6- and 12-month evaluations was an increase in the number of marrow hematopoietic progenitor cells. The researchers conclude, "The experiments reported here demonstrate alterations in the hematopoietic and immunologic systems of rats after long-term exposure (6 and 12 mo) to very low levels of RFR (SAR, 0.15–0.4 W/kg; .48 mW/cm^2)" (Chou et al. 1985).

A study at Georgia Tech examined the effects of pulsed RF (PRF = 1 kHz, width = 1 μs) on 100 Sprague-Dawley rats (100 controls), to simulate exposure of human beings to 50 MHz. No differences were found in a large number of bloodborne end points. Levels of the neurotransmitter, dopamine, were lower in the exposed group than in the control group. According to the researchers, "Though significant, this small decrease might not be physiologically important" (Bonasera, Toler, and Popovic 1988; Toler et al. 1988).

EPA researchers studied the effect of long-term exposure (2450 MHz and either 2 or 6.8 W/kg) on the life span of CD1 mice. In comparison to sham controls the high exposure group had a statistically significant shorter average life span, 572 days versus 706 days. The low SAR group had a non-significantly longer life span than controls (Liddle, Putnam, and Huey 1994).

In summary, chronic effects have been examined in mice, rats, rabbits, and dogs exposed to RF between 50 MHz and 24 GHz, with 2450 MHz being predominant. SARs ranged from 0.15 W/kg to around 50 W/kg, and durations were from 40 days up to 25 months. Many studies used small groups of animals, thereby limiting the utility of the results (Deichmann et al. 1963; Ferri and Hagan 1977; Guy et al. 1980; McRee et al. 1980). Most studies used one sex of a single species, exposed at one dose rate. The most consistent results are effects on the hematopoietic and immune systems at exposure levels (SARs = 0.15–0.4, 1.5, 2.2 W/kg) that do not produce measurable signs of stress. Generally, however, the effects were not observed on the same end point and were not reproducible. For example, in studies that exposed rabbits at the same frequency and similar SARs, Ferri and Hagan (1977) found differences in RBC count, while Guy et al. (1980) found a nonsignificant reduction in WBC count and a significant, reversible reduction in eosinophils, but no differences in RBC count. A stimulatory effect on the immune system was seen at 13 months into a study, but not at 25 months (Guy et al. 1985). The 13-month effect was not replicated in a separate study, although an effect on hematopoietic cells was observed (Chou et al. 1985). Dumanskij and Sandala (1974) found effects on levels of cholinesterase, sulfhydryls, and 17-ketosteroid varied, but this was not independently replicated (D'Andrea et al. 1986a, 1986b).

The possible reasons for these discrepancies may be due to methodologic differences, sampling errors, or artifacts. As has been pointed out, it is possible that some biologic differences between groups of animals may be expected due to chance in studies that look at a large number of end points (McRee et al. 1980).

3.3.2 Ocular Effects

Effects have been demonstrated on the cornea, iris vasculature, lens, and retina. The results of

Table 3-3. Ocular Effects

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Rabbits (FF)	468 CW	8.1 ± 1.2	60	10×20	No ocular	Cogan
	385 CW	24[a]	30	10×90	effects;	et al.
	385 CW	48[a]	60	10×15	death in	1958
		WBA			some cases	
Rabbits (NF)	5500 CW	325 to 525[b]	47	1×1.5	Cataract threshold	Birenbaum 1969a
	5500 Pulsed	325 to 500[b]	78.5[a]	1×125		Birenbaum 1969b
	800 CW	500[b]	78.5[a]	1×25	Cataracts	
			CORNEA AND IRIS			
Monkeys (FF)	9310 Pulsed	NR	150	30 or 40 × 294 or 665	No cataracts or corneal lesions	McAfee et al. 1979
Rabbit (FF)	2450 CW 2860 Pulsed	NR	225	1×10 up to 25×30	No effects on normal corneas or healing of wounded corneas	Williams and Finch 1974
Mouse	34,000 CW	NR	0.02	10×1020	No effects found by light and electron microscopy; DNA synthesis decreased	Rotkovska et al. 1993
Monkey	2450 CW	1.3	5	44×240	No effects on cornea;	Kues et al. 1985
		2.6	10	10×240	no effects;	
		2.6	10	56×240	minor changes;	
		5.2	20	18×240	no effects;	
		5.2	20	22×240	minor changes;	
		7.8	30	5×240	moderate change;	
		7.8	30	8×240	moderate change;	
	2450 Pulsed	1.3	5	5×240	no effects on cornea;	
		2.6	10	4×240	minor to major changes;	
		3.9	15	1×240	minor to major changes	

Table 3-3. (Continued)

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Monkey	2450 Pulsed	2.65	10	3×240	Damage to corneal endothelium; increased iris vascular permeability	Kues and D'Anna 1987
Rabbit	35,000 Pulsed	33 (avg.)	23.4	1×15	Single-cell destruction in cornea; increasing corneal effects	Trevithick et al. 1987
		109, 550, 3276	78, 390, 2340	1×15		
			LENS			
Rabbits (NF)	2450 CW	60 to 200[b]	80 to 400	1×5 to 1×60	Cataract threshold	Carpenter and Van Ummersen 1968
	2450 CW	140[b]	280	4×4	Opacities observed; cumulative effects from subthreshold doses; no cataracts	
				1×5		
		60[b]	120	5×25		
				3×30		
		40[b]	80	10×60		
				15×60		
		20[b]	40	15×60		
Rabbits (NF)	2450 CW	138	150	1×100	Cataract threshold	Guy et al. 1975b
		184	200	1×20		
		460	500	1×5		
		Local maximum				
Rabbits	3000 Pulsed	14,[a] WBA	100	1×15 or 1×30	No cataracts; acute as iritis; death at 30 min + 300 mW/cm^2 and at 500 mW/cm^2	Appleton, Hirsch, and Brown 1975
		28	200			
		42	300			
		56	400			
		70	500			
Rabbits (NF)	2450 CW	15.3	NR	1×30	Cataract incidence in 50% of eyes; threshold estimated	Foster, Ferri, and Hagan 1986

Table 3-3. (Continued)

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Rabbits (FF)	3000 CW	14, 28[a]	100, 200	1×15 or 1×30	No ocular changes	Appleton 1974a
Rabbits (FF)	2450 CW	1.5[a]	10	5×480 8 to 17 weeks	No cataracts	Ferri and Hagan 1977
Rabbits (FF)	2450 CW	17 (maximum WBA)	10	180×1380	No ocular effects	Guy et al. 1980
Rat lenses[c]	915 Pulsed	120 (37°C)	NR	1×0.5	Holes in lens cells; large globules indicative of much higher temperature elevation	Stewart-DeHaan et al. 1983
		400 (39°C)	NR			
		1200 (37°C)	NR			
Rat lenses[c]	1250 CW 1250 Pulsed	9.2 1.9	NR NR	1×6, 20, 60	Damage threshold estimated	Bassen et al. 1987
Rat lenses[c]	918 Pulsed	20, 40 10	NR NR	1×6 1×60	Lens fiber effects	Stewart-DeHaan et al. 1985
Dogs	24,000 Pulsed 1285 Pulsed	NR 1, 2.5, 5[c]	24 20, 50 100	600×990 1×360 10×360 20×360	No ocular effects	Michaelson, Howland, and Deichmann 1971
Rabbits (NF)	2450 CW	460[d]	295	1×30	Cataracts 50% of eyes; cataracts in all eyes exposed	Hagan and Carpenter 1976
	10,000 CW	590[d]	375	1×30		
	2450 CW	510[d]	325	1×30		
	10,000 CW	640[d]	410	1×30		

[a]Estimate from Elder and Cahill (1984).
[b]Estimate for 2-g eye.
[c]Estimated whole-body SAR from Durney, Massoudi, and Iskander 1986.
[d]Estimate for 2-cm diameter area for 2-g eye.
[e]In vitro experiment.
(FF), far-field conditions; (NF), near-field conditions; CW, continuous wave; WBA, whole-body average; NR, not reported.

selected studies are compiled in Table 3-3. Except for studies of the lens, most of these effects have been established in a single species and in one laboratory and, hence, cannot be viewed as conclusive.

As a first approximation, the penetration depth of RF radiation varies inversely with frequency. For ocular tissues, this was demonstrated at 70 GHz (Birenbaum et al. 1969a) and at 35 and 107 GHz (Rosenthal et al. 1976). At 70 GHz, effects were greatest at the cornea, with diminishing effects to deeper ocular structures such as the conjunctiva, iris, and lens. As the wavelength lengthens, the penetration depth increases so that the lens becomes the critical ocular structure at frequencies < 10 GHz. At frequencies less than around 800 MHz, ocular structures have not been implicated as being at primary risk. Numerical calculations using a 45,024-cell model indicate that the highest part-body SARs above 200 MHz are for the eyes and that the SAR increases at frequencies greater than 350 MHz because of "the superficial nature of EM deposition at higher frequencies" (Gandhi et al. 1992).

3.3.2.1 *Cornea*

McAfee et al. (1979) trained monkeys to face into a horn antenna and press a lever to receive apple juice through a tube. Pressing the lever activated pulsed 9.3-GHz microwaves. No corneal lesions were observed with slit-lamp biomicroscopy. No dosimetry was reported. Williams and Finch (1974) exposed rabbits to 2.86-GHz pulsed and 2.45-GHz CW microwaves at power densities of 225 mW/cm^2, and exposure durations of 10–30 min/d for up to 5 weeks. No effects were detected in normal or wounded tissues by either histologic evaluation or autoradiography. Rotkovska et al. (1993) found no corneal damage in hairless mice exposed at 34 GHz and 20 μW/cm^2. DNA synthesis in corneal cells was nonsignificantly reduced in the exposed animals.

Effects on the corneal endothelium were seen in studies at Johns Hopkins University (Kues et al. 1985; Kues and D'Anna 1987). In humans, the endothelial layer is composed of single cells that do not regenerate, which must be factored into the selection of a suitable animal model. The study design at Johns Hopkins used cynomolgus monkeys, a species in which the endothelial cells do not regenerate. Ocular-region SARs were estimated to be 1.3 to 7.8 W/kg. Examinations included specular microscopy, histologic staining, and transmission electron microscopy. The results showed insignificant temperature rise, cellular lesions and cell death, and areas of enlarged cells and areas lacking cells. A latency period of 16 hours or more was found before effects were observed; 72 to 96 hours after exposure the appearance of the endothelium was normal, but the researchers report "large cells in areas of previous microwave damage suggesting a loss of cells" (Kues et al. 1985). Pulsed MW produced abnormalities at lower-power densities than CW exposures, but predominant effects were seen at the 30 mW/cm^2 level. Kues et al. (1992) report that pretreatment with two ophthalmic drugs, timolol maleate and pilocarpine, enhanced effects on the corneal endothelium.

Typically, SARs were not estimated in studies performed prior to about 1980, so SARs were not reported in two of the studies referenced above (McAfee et al. 1979; Williams and Finch 1974). Also, exposure durations were controlled by the animals in the operant response experiment using apple juice where total exposure time varied from 294 to 665 minutes (McAfee et al. 1979). The exposure durations in the other studies are not comparable. Average power densities ranged from 5 to 225 mW/cm^2. Hence, it is difficult to determine why corneal damage was observed at lower dose rates in one study (Kues et al. 1985) than in the others. It is possible that the differences in results may be attributable to methodologic differences, i.e., species selection and examinations.

The corneal epithelial tissues of New Zealand white rabbits were exposed to CW and pulsed, 35-GHz MW. Peak power density was 15 kW/cm^2, and time-averaged values are shown in Table 3-3. SARs were 1.4 W/kg/mW/cm^2. Microscopic examination showed cell damage, craterlike spots, and coagulation. Damage was noted at the lowest time-averaged SAR, 33 W/kg, from a 15-minute exposure at a time-averaged power density of 23 mW/cm^2 (Trevithick et al. 1987).

3.3.2.2 *Iris*

In an extension of their work with the endothelium, Johns Hopkins researchers evaluated the iris of cynomolgus and rhesus monkeys. Animals were exposed to 2.45 GHz, pulsed emissions with a local SAR of approximately 2.65 W/kg. The results showed increased iris permeability (blood-aqueous barrier) to a tracer molecule, and endothelial lesions. A correlation was found between increased iris permeability and subsequent development and severity of effects on the endothelium (Kues and D'Anna 1987). Pretreatment with two drugs used in the treatment of glaucoma, timolol maleate and pilocarpine, enhanced the effects (Kues and Monahan 1992; Kues et al. 1992).

3.3.2.3 *Lens*

The lens appears to be at high risk of microwave-induced damage due to its avascular nature. The initial works evaluating the potential for lenticular changes were published in the late 1940s (Daily et al. 1948; Richardson, Duane, and Hines 1948). Since that time, both in vivo (Daily et al. 1948; Richardson, Duane, and Hines 1948; Williams et al. 1955; Cogan et al. 1958; Carpenter and Van Ummersen 1968; Birenbaum et al. 1969a, 1969b; Carpenter 1975; Guy et al. 1975b, 1980; Hagan and Carpenter 1976; Emery et al. 1975; Ferri and Hagan 1977; Hirsch et al. 1977; McAfee et al. 1979; Foster, Ferri, and Hagan 1986; Kramar, Harris, and Guy 1987) and in vitro studies have been performed (Stewart-DeHaan et al. 1983, 1985; Steel and Sheppard 1986; Bassen et al. 1987). Reviews of MW cataractogenesis are available (Seth and Michaelson 1965; Milroy and Michaelson 1972a; Cleary 1980; Elder and Cahill 1984). The following is a summary of the published information.

Rabbits, monkeys, and dogs were exposed at frequencies between 385 MHz and 70 GHz. Both CW and pulsed fields were used, and animals were located both in the near and far fields. No cataracts were found when unrestrained animals received far-field exposures (Michaelson, Howland, and Deichmann 1971), even when the exposures were nearly lethal (Appleton, Hirsch, and Brown 1975). The most effective frequencies were 1 to 10 GHz, and acute thresholds for cataract formation have been established (Carpenter and Van Ummersen 1968; Guy et al. 1975b). For a single exposure, a 150 mW/cm^2, 100-minute exposure threshold was established. (Guy et al. 1975b). Figure 3-2 shows the threshold curve for maximal SAR and exposure duration. No cataracts were seen at 200 mW/cm^2 for 30 minutes (Appleton, Hirsch, and Brown 1975). Foster, Ferri, and Hagan (1986) reported the dose rate effective in producing a 50% incidence of cataracts in rabbits as 15.3 W/kg to the animal's head. In other studies, SARs varied between 100 to 500 W/kg in the eye and 1.5 to 70 W/kg for the whole body. A latency period of 24 to 48 hours was observed. At least one study found cumulative effects from subthreshold exposures (Carpenter and Van Ummersen 1968), although another researcher did not come to this conclusion (Appleton 1974a). Chronic exposures have not produced cataracts in test animals (Deichmann et al. 1963; Ferri and Hagan 1977; Guy et al. 1980). Age of the test animal does not appear to be an important factor in MW-induced cataracts (Van Ummersen and Cogan 1965). Absorption and heating patterns seem dependent upon facial characteristics and wavelength. Clearly, temperature elevation can produce a thermal cataract (Kramar, Harris, and Guy 1987; Emery et al. 1975; Guy et al. 1975b), but some characteristics of microwave exposure may also produce cataracts, although this requires further definition (Carpenter and Van Ummersen 1968; Stewart-DeHaan et al. 1983; Bassen et al. 1987). Some reports suggest that it is more likely to observe MW-

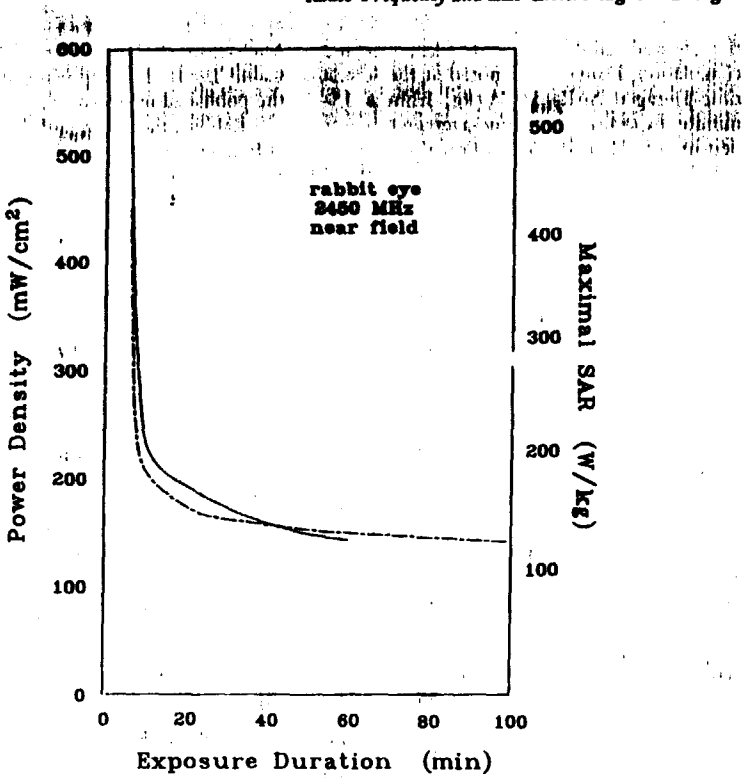

Figure 3-2. Cataractogenic threshold for the rabbit eye for near-field exposures at 2450 MHz. After Guy et al. (1975b).

induced cataracts on the posterior capsule of the lens (Guy et al. 1975b; Zaret 1972), although this is not a consensus opinion (Tengroth 1983; Silverman 1985). Seth and Michaelson (1965) have suggested that the appearance of some small, microwave-induced lenticular opacifications may be a reversible phenomenon.

In vitro studies with rat lenses show physical damage including small holes, foam, large globules, and capsular damage. Local SARs were determined (Table 3-3), and lenticular temperature was carefully controlled by a circulating buffer solution. At a given temperature, the amount of damage was roughly proportional to the SAR. An evaluation of the effects and the measured temperatures led the authors to conclude that the effects were due to microwave exposure and not increased temperature (Stewart-DeHaan et al. 1983). Examination by scanning electron microscopy found effects on lens fiber cells after 6-minute exposures at local SARs of 20 and 40 W/kg and after a 1-hour exposure at 10 W/kg. Time-dose rate reciprocity was suggested (Stewart-DeHaan et al. 1985). Pulsed MW exposure induced cataracts at lower dose rates than CW exposures in one in vitro study. Bassen and colleagues (1987) report lens damage with pulsed radiation at a time-averaged, lenticular SAR of 1.9 W/kg, versus 9.2 W/kg for CW exposure.

3.3.2.4 Retina

Reports have been published claiming retinal damage in rabbits and primates. Rabbits received far-field exposures to the right side of the head at 3.1-GHz pulsed MW and an average power density of 55 to 57 mW/cm^2. No cataracts were observed. In five animals examined for retinal histology by electron microscopy, all exhibited some level of change in retinal plexiform layers (Paulsson et al. 1979). Restrained, unanesthetized primates were exposed to pulsed microwaves (1.25 GHz, width = 0.5 μs, PRF = 16 Hz, 1 megawatt peak power) at 4 W/kg for nine 4-hour treatments. Changes in photoreceptor responses were reported when pretest and posttest values were compared. Histopathology indicated "degenerative changes" of the photoreceptors (Kues and Monahan 1992).

3.3.3 Auditory Phenomenon

The auditory phenomenon, or microwave hearing, has been established in both humans (Frey 1961; Sommer and von Gierke 1964) and laboratory animals (Chou, Guy, and Galambos 1982; Chou, Yee, and Guy 1985). Literature reviews are available (Lin 1980; Chou, Guy, and Galambos 1982; Elder and Cahill 1984). Justesen (1975) has written an interesting treatise that discusses the auditory phenomenon and includes unpublished references to human communication via telegraphy and voice-modulated MW.

The frequency range that stimulates the response is around 200 MHz to 8 GHz. The exposures are pulsed at a PRF between 0.5 and 1000 Hz, with pulse widths between 1 and 1000 seconds. Exposures to CW fields do not produce auditory effects. Whole-body values of specific absorption in rats were 0.9 to 1.8 mJ/kg/pulse (Chou, Yee, and Guy 1985). The threshold for audibility in humans at 2450 MHz is around 400 mJ/m^2 when the pulse is less than 30 μs (Guy et al. 1975a; Lin 1989).

More recent work has examined the auditory response to pulsed RF generated by coils used in magnetic resonance imaging systems, at frequencies between 2.4 and 170 MHz. With the head within the coil and pulse widths from about 3 μs to 100 μs, the threshold energy was 16 ± 4 mJ/pulse. If the coil was located at the ear, threshold energy as low as 3 mJ produced the sensation in human volunteers (Roschmann 1991).

The effects manifest as audible clicking, hissing, buzzing, chirping, and popping sounds that seem to originate from behind or within the head. The mechanism is believed to be thermoelastic expansion within the head. The ensuing pressure wave "is detected by the hair cells in the cochlea via bone conduction" (Elder and Cahill 1984). The auditory phenomenon has not been shown to be adverse, although Lin (1989) cautions that the question of health risk from exposure to RF pulses at power levels well above threshold has not been answered.

3.3.4 Nervous System

Effects studied include influences on the brain and behavior, calcium efflux from brain tissues, changes in the permeability of the blood-brain barrier, electroencephalographic changes, brain energy metabolism, interaction with psychoactive drugs, and effects on neurotransmitters. Neuroendocrine effects will be addressed separately. The results of some studies are compiled in Table 3-4.

3.3.4.1 Brain

No differences were found in the temperature in various sections of the brain in anesthetized rats exposed to either microwaves or elevated ambient temperatures. SAR measurements indicated that energy absorption was lowest in the cortex and highest in the olfactory lobes. In a second study, the temperature rise in the cortex was more rapid initially with exposure to MW energy, but within 5 minutes, all differences had disappeared. No hot spots were observed (Ward et al. 1986).

Baranski found lesions in the brain and cerebellum and glial cell proliferation. These effects were found for both CW and pulsed exposures, but pulse-induced effects were more

Table 3-4. Selected Nervous System Effects

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Rats	2450	0, 2, 4, 6	0, 10, 20, 30	1×30	Variable energy absorption by brain area	Ward, et al. 1986
	2450	6	30	1×30	Initial difference in temperature rise in cortex	
Rabbits	3000 CW and pulsed	0.4[a]	3.5	90×180	Brain lesions and glial cell proliferation	Baranski 1972
Guinea pigs	3000 CW and pulsed	0.5[a]	5	90×180		
Rats	3000 Pulsed	1.0[b]	5	10 d	Neurons synchronized to PRF	Servantie, Servantie, and Etienne 1975
Rabbit	2400	7.2[b,c]	40	1×1	Changed neuronal discharge frequency	Chizhenkova 1988
Rabbits	2950 Pulsed	1.0[b]	5	90×120 120×120	Changes to EEG	Baranski and Edelwejn 1975
		1–6[b]	5 to 30	1×120	Slight effects at 30 mW/cm^2	
Monkeys	388	NR	NR	1×3 or 1×15	Arousal, drowsiness, convulsions, EEG changes	Baldwin, Bach, and Lewis 1960
Cats	147 AM	0.015[a]	<1	Various	Changes in EEG	Bawin, Gavalas-Medici, and Adey 1973
Rabbits	2375 ±50 CW	0.002[b]	0.01	NR	Alpha rhythm intensified; depression of bioelectric activity	Shandala, Rudnev, and Los 1981
		0.09[b]	5	NR		
Monkeys	2450 Pulsed	0.0034, 0.34, 3.4	NR	295×210 to 335×210[d]	No differences in EEG	Kaplan et al. 1982
Hamsters	1700	3.0[a]	1	1×30 or 1×120	Altered neurons	Albert 1977
	1700	7.5[b]	2.5	1×30 or 1×120		

Table 3-4. (Continued)

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Chicken[e]	147 AM: 0.5 to 35 Hz	0.002[a]	1	1×20	Increased Ca^{+2} efflux at specific AM frequencies	Bawin, Kaczmarek, and Adey 1975
Chicken[e]	147 AM: 3 to 30 Hz	0.0014[a]	0.5–2	1×20	Increased Ca^{+2} efflux	Blackman et al. 1979
Neuro-blastoma cells[f]	915 AM: 16 Hz	0.05, 0.75 1.0	NR	1×30	Increased Ca^{+2} efflux	Dutta et al. 1984
Neuro-blastoma cells[f]	147 AM: 16 Hz	0.05, 0.005	NR	1×30	Increased Ca^{+2} efflux	Dutta et al. 1989
Rat[g]	1000 Pulsed PRF = 16 Hz	0.29, 2.9	1, 10	1×20	No significant differences in efflux	Merritt, Shelton, and Chamness 1982
	2450 Pulsed	0.3	1	1×20	No significant differences in Ca^{+2} efflux	
Rat	2060 CW and pulsed PRF = 8, 16, and 32 Hz	0.12, 0.24 1.2, 2.4	0.5, 1, 5, 10	1×20	No significant differences in efflux	Merritt, Shelton, and Chamness 1982
Cats	450 AM: 16 Hz	0.29	3	1×60	Increased Ca^{+2} efflux	Adey, Bawin, and Lawrence 1982
Rats (male)	1300 CW and pulsed	0.01 to 0.4[b]	0.03 to 2	1×20	Increased permeability to mannitol and inulin	Oscar and Hawkins 1977
Rats	2450	0.02 to 6[b]	0.1 to 30	1×30	No differences in mannitol permeability	Preston, Vavasour, and Assenheim 1979
Rats	2450	0.04 to 200	0.5 to 2600	1×20	No differences in Evans blue permeability;	Lin and Lin 1982
	2450	240	3000	1×20	Increased permeability to dye; hyperthermia	Lin and Lin 1982

Table 3-4. (Continued)

Species	Frequency (MHz)	SAR (W/kg)	Average Power Density (mW/cm^2)	Duration (d×min)	Effects	Reference(s)
Rats	2450	NR	3000	1×15	Increased permeability to dye; inversely related to ethanol concentration	Neilly and Lin 1986
Rats	3000 Pulsed	1[a]	4 to 6	10–15 d	Reduced effect of paralyzing drugs	Servantie et al. 1974
Mice	2450 CW	45	NR	1x/wk	Changes in escape avoidance behavior	Monahan and Henton 1979
Rabbits	2450 CW and pulsed	0.9, 1.8, 2.7, 9	5, 10 25, 50	NR	Reduced sleeping times	Wangemann and Cleary 1976
Rats	2450 Pulsed	0.6	1	1×45	Orientation-dependent differences in recovery from hypothermia	Lai et al. 1984a
Rats	2450 CW	0.3	NR	1×45	Attenuation of ethanol-induced hypothermia in rats exposed to MW; effect not seen in rats treated with neurotoxin	Hjeresen, Francendese, and O'Donnell 1989
Rats	2800 Pulsed	1.9[b]	10	1×10	Decrease in body temperature	Ashani, Henry, and Catravas 1980

[a]Estimate from Elder and Cahill (1984).
[b]Estimate based on Durney, Massoudi, and Iskander (1986).
[c]Whole-body average.
[d]The number of exposure sessions are estimated from the range of gestational age and the assumption of a full year of exposure.
[e]In vitro experiment.
PRF, Pulse repetition frequency; CW, continuous wave; AM, amplitude modulated; NR, not reported; EEG, electroencephalogram.

intense (Baranski 1972). Albert and Desantis (1976) found no effects on glial cells in Chinese hamsters, but hypothalamic and subthalamic neurons in the exposed animals had more cytologic alterations than controls.

Studies of the effects of RF fields on brain energy metabolism have been performed using three biochemicals as markers: nicotinamide adenine dinucleotide (NADH), adenosine triphosphate (ATP), and creatine phosphate (CP) (Sanders, Schaefer, and Joines 1980; Sanders and Joines 1984; Sanders, Joines, and Allis 1984, 1985). The results are summarized in Table 3-5. The 591-MHz field was also amplitude modulated at several frequencies between 4 and 32 Hz, and pulse-modulated. The authors hypothesize that the observed effects are not thermal and that RF fields directly inhibit mitochondrial energy production pathways (Sanders and Joines 1983; Sanders and Joines 1984; Sanders, Joines, and Allis 1985). However, the results were not consistent across the three frequencies.

Western and Eastern researchers evaluated behavioral, biochemical, and electrophysiologic parameters in duplicate projects. Prior to exposure, male rats were allowed to adapt to environmental conditions, then exposed in small groups for a single 7-hour period at WBA-SARs of 2.7 W/kg (2450 MHz and 10 mW/cm^2). Biochemical measurements from cerebral cortex samples included Na^+, K^+, Mg^{2+}, and Ca^{2+} ATPase, and K^+-alkaline phosphatase. One group found a statistically significant depression in Na^+ and K^+-ATPase activity in the MW-exposed animals, but this was not observed by the other group. The authors note that the effects may be spurious or due to methodologic differences in biochemical techniques (Mitchell et al. 1989).

3.3.4.2 *Electroencephalogram (EEG)*

The effect of RF fields on the EEG have been studied (Table 3-4). RF exposure synchronized the cortical neurons in rats at the PRF of the exposure field (Servantie, Servantie, and Etienne 1975). Increases and decreases in the neuronal discharge frequency occurred in unanesthetized rabbits exposed for 1 minute at 2.4 GHz and 40 mW/cm^2 (Chizhenkova 1988). No effects on the EEG were found from a single exposure to rabbits, except at the highest power density, 30 mW/cm^2. Repeated exposures desynchronized the EEG, and effects were more pronounced with pulsed fields (Baranski and Edelwejn 1975). Observations were made of clinical signs in monkeys exposed to the 100-W output of a 388-MHz transmitter. Whole-body exposures produced no effects, while exposure of just the head affected the EEG (Baldwin, Bach, and Lewis 1960). Cats were exposed to a 147-MHz carrier wave that was amplitude modulated between 1 and 25 Hz, with modulation percentage up to 90%. When modulation frequencies were close to naturally occurring frequencies, there was a higher incidence of changes in the EEG (Bawin, Gavalas-Medici, and Adey 1973). Shandala and

Table 3-5. Results of Studies on Brain Energy Metabolism in Rats

Frequency (MHz)	Head SAR (W/kg)	NADH Fluorescence[a]	ATP[b]	CP[b]
200	0.02–1.84	Dose-dependent increase	Decreased	No change
591	0.09–7.40	Dose-dependent increase	Decreased	Decreased
2450	0.18–14.72	No change	No change	No change

[a]Measured with anesthesized rats.
[b]Measured after sacrifice.

colleagues reported EEG changes in rabbits as noted in Table 3-4 (Shandala, Rudnev, and Los 1981).

Pregnant squirrel monkeys were exposed to MW from the second trimester to 12 months after delivery. SARs in Table 3-4 are for adult monkeys at PRF = 60 Hz. No differences were observed in maternal EEG, infant EEG at 6 months, or in infant EEG at the lowest dose rates at 9 and 12 months. High infant mortality prevented researchers from having sufficient animals to perform a meaningful test at 9 and 12 months for the high-dose-rate group (Kaplan et al. 1982).

Johnson and Guy (1972) have concluded that it is possible that the use of metallic electrodes and conductive leads (Michaelson 1982a) may have compromised the utility of some of the early EEG studies. Modern experiments use nonconductive materials such as glass electrodes and polyvinyl-chloride leads to minimize perturbations.

3.3.4.3 *Calcium Efflux*

Calcium is an important ion in many bodily mechanisms (Case 1980). Doubly ionized calcium (Ca^{+2}) has been used as a marker of the potential for RF-induced nervous system effects, primarily in in vitro experiments. Typically, the experimental design involves extraction of brain tissue from neonatal chicks. The brain is divided and placed in a physiologic medium that contains radioactive calcium ($^{45}Ca^{+2}$). Following a labeling period, the tissues are rinsed and transferred to tubes containing fresh physiologic medium, then exposed to an amplitude-modulated RF carrier wave. Typically, modulating signals are in the ELF or sub-ELF bands. Aliquots of the physiologic solution are collected after exposure and assayed for radioactivity. If the physiologic solution containing the RF-exposed brain hemispheres contains more ^{45}Ca, the conclusion is that there has been calcium efflux.

Studies of Ca^{+2} efflux have demonstrated some remarkable findings, as reviewed in detail in other publications (Adey 1980; Liddle and Blackman 1984; Postow and Swicord 1986; NCRP 1986; Blackman 1990). Foremost among the findings is that the effects occur at very low SARs, between 0.0005 and 2.9 W/kg (Elder and Cahill 1984). Calcium efflux demonstrates specificity, or windowing, as a function of carrier frequency, modulating signal frequency, power density, and temperature. This means that some exposures are effective in producing the effect, while others are not, and this does not appear to be dose dependent. RF carrier frequencies that are effective include 50, 147, 450, and 915 MHz. Modulating signal frequencies of 6, 9, 11, 16, 20, or 32 Hz have been effective, while other frequencies have not. For example, Bawin, Kaczmarek, and Adey (1975) exposed brain tissues to both a modulated and unmodulated 147-MHz carrier wave. Modulation depths were between 80 and 90%. Statistically significant differences between exposed and control tissues were found at modulation frequencies of 6, 9, 11, 16, and 20 Hz. Ineffective frequencies were 0.5, 3, 25, and 35 Hz, and the unmodulated, 147-MHz carrier.

Bawin and Adey (1977) report an increase in efflux at 1 mW/cm^2, but no differences when the intensity was doubled. They also found a narrow amplitude window for ELF E-field exposures between 10 and 100 V/m (Adey and Bawin 1982). EPA researchers studied windowing effects as a function of power density (Blackman et al. 1979). Brain tissues were exposed to a 147-MHz carrier with a modulation frequency of 16 Hz. (Some studies have shown that 16 Hz is the most effective AM frequency for producing Ca efflux.) Power densities were 0.5, 0.75, 1, 1.5, and 2 mW/cm^2. A statistically significant increase was observed at 0.75 mW/cm^2. Nonsignificant increases were seen at 0.5 and 2 mW/cm^2, while decreases were found at other intensities. Blackman and colleagues (1989) extended this work using a 50-MHz carrier, modulated at 16 Hz. The SAR was 0.36 $mW/kg/mW/cm^2$, with the highest value estimated at 0.005 W/kg at a power density of 14.7 mW/cm^2. Enhanced efflux was found at 1.75, 3.85, 5.57, 6.82, 7.65, 7.77, and 8.82 mW/cm^2. No differences between sham and exposed groups were seen at 0.75, 2.3, 4.5, 5.85, 7.08, 8.19, 8.66, 10.6, and 14.7 mW/cm^2.

Human neuroblastoma cells were exposed at 915 MHz, with either unmodulated or modulated (16-Hz, modulation depth = 80%) fields. SARs for culture medium and cells were 0.01, 0.05, 0.075, 0.1, 0.5, 0.75, 1.0, 1.5, 2, or 5 W/kg. SARs for neuroblastoma cells were not determined. For an unmodulated 915-MHz carrier, efflux was significantly different for SAR = 1.0 W/kg but not for a SAR of 0.05 W/kg. The authors conclude that a narrow range of effective deposited powers exists, and the effect does not appear to be thermal in nature (Dutta et al. 1984). In an extension of this work, Dutta and colleagues (1989) evaluated efflux of avian, feline, Chinese hamster-mouse, and human neuroblastoma cells, with findings similar to those described earlier.

Rat brain tissue was exposed to either 1- or 2.45-GHz pulse-modulated MW, at a PRF of 16 or 32 Hz. No difference was seen in efflux of Ca at any of the SARs (Table 3-4). In one set of experiments brain tissue was removed and placed in a radioactive solution for a 20-minute labeling period as discussed earlier (Shelton and Merritt 1981). In a second study, ^{45}Ca was introduced into the brain tissue via intraventricular injection into intact rats. The animals were euthanized and the brains immediately removed and placed in physiologic solution prior to exposure (Merritt, Shelton, and Chamness 1982). The differences in experimental outcome between these experiments and those reported earlier may be attributed to methodologic differences. It is possible that the pulse-modulated signal may not be biologically demodulated in a manner similar to an AM wave, although the interaction mechanism(s) are not well understood (Shelton and Merritt 1981).

In 1985, EPA workers suggested that the earth's local geomagnetic field could be an experimental variable in efflux research. This conclusion developed from the observation of differences in efflux in laboratories located in North Carolina and California. Experiments at the EPA laboratory in NC had demonstrated enhanced efflux, while in CA, Bawin observed a reduced flux. EPA experiments used an electromagnetic field, while Bawin used just an E field (Blackman et al. 1985). This seemed to imply a possible role for the time-varying magnetic field, so an experiment was designed to evaluate differences in the direction of Ca mobility by exposing tissues to either an electromagnetic (40 V/m and 59.5 nT) or an E field. (The E-field study actually included a very weak magnetic flux density, 15 pT, and a 40-V/m E field.) The experiment was performed within a Helmholtz coil, so the local geomagnetic flux density could be controlled. A significant difference in the outcome was seen when an electric versus an electromagnetic field was used. Windowing was reported, but the modulation frequencies that were not effective in producing efflux could be made effective by changing the flux density of the local geomagnetic field. Conversely, effective frequencies could be rendered ineffective, again by varying the local geomagnetic field (Blackman et al. 1985).

Further work showed that the thermal history of the tissues during experimental preparation and exposure is critically important, demonstrating a "temperature window." Hence, the use of an appropriate temperature is "necessary to establish a consistent response from the brain-tissue preparation" (Blackman, Benane, and House 1991).

Few in vivo experiments have been performed. Female cats were exposed as shown in Table 3-4, at a modulation depth of 85%. "By comparison with controls, efflux curves from field exposed brains were disrupted by waves of increased $^{45}Ca^{2+}$ efflux. These waves were irregular in amplitude and duration, but many exhibited periods of 20–30 min" (Adey, Bawin, and Lawrence 1982). In another in vivo experiment, radioactive Ca^{+2} was injected intraventricularly into male rats 2 hours prior to 20-minute exposure to either CW or pulsed fields at 2060 MHz. No significant differences were found (Merritt, Shelton, and Chamness 1982).

The significance, and in some cases the very existence, of Ca^{+2} efflux as a biologic effect has been disputed. This is because the results are not robust and have not been consistently replicated. It should be apparent from the previous review that efflux is highly de-

pendent upon methodology. Indeed, the lack of replication may be due to methodologic differences, as pointed out by Blackman, Benane, and House (1991). Some of the differences include the length of the labeling period, activity of the labeling solution, sample temperature, volume of the physiologic solution, type of modulation, and modulating frequency. The viability of the tissues themselves has been questioned. One of the principal researchers has observed that the brain tissues used in the studies are electrically dead but are still able to metabolize oxygen (Blackman 1990).

This particular effect also has another dimension: It has become the fulcrum in a debate over thermal and nonthermal effects. This debate has manifested a spectrum of ideas. At one end are those who believe that observation of calcium efflux is an artifact of poor experimental technique. Opposing this are those who believe that calcium-efflux experiments will help elucidate a better fundamental understanding of the operation of the nervous system. These differences will probably only be reconciled by well-conceived, -designed, and -implemented in vivo experiments. These experiments must evaluate biologic end points that are generally recognized as RF-induced and have a testable hypothesis involving calcium. One such possible avenue of research is being undertaken where scientists are evaluating the effect of electromagnetic energy and calcium, using calcium chelating agents, on short-term behavior (Anderson 1990). Although no definitive conclusions have been reached at this writing, it is this type of experiment that will help determine if calcium efflux is an interesting biologic novelty or the portend of a potential hazard.

3.3.4.4 *Blood-Brain Barrier*

Physiologically, this barrier is the interface between the brain and the blood. The barrier acts like a filter to high-molecular-weight substances, a fact that has been used in studies of changes in permeability of the blood-brain barrier (BBB).

A significant increase in fluorescence of brain sections of rats anesthetized with sodium pentobarbital has been observed (Frey, Feld, and Frey, 1975). Albert (1977) observed horseradish peroxidase leakage in brain tissue of both controls and exposed Chinese hamsters (2450 MHz, 10 mW/cm^2). Albert and Kerns (1981) detected lesions at 2.5 W/kg. Animals that were exposed and allowed a 2-hour recovery period had no gross lesions, demonstrating a reversibility of the effect.

Oscar and Hawkins (1977) reported a statistically significant increase in permeability to radio-labeled mannitol and inulin, but not to dextran, in rats that were anesthetized with pentobarbital. However, Preston and coworkers were unable to replicate this finding. In a discussion of these differences, they suggest that the observations made by Oscar and Hawkins may be due to variations in blood flow that were not accounted for in the experimental methods (Preston, Vavasour, and Assenheim 1979). Oscar et al (1981) examined this premise and stated that their findings "must be re-evaluated in light of these observed changes in blood flow."

It appears that BBB effects are associated with local hyperthermia. Measurement of brain temperature during exposure of the heads of anesthetized rats at 2450 MHz showed that permeability of the BBB to horseradish peroxidase was effected by MW-induced hyperthermia (Sutton and Carroll 1979). Extreme hyperthermia altered the BBB in rats. No effects were seen when exposures did not thermally stress the animals (Lin and Lin 1982). Studies at 1.2 and 1.3 GHz indicated that the rat brain must be made hyperthermic before changes in permeability were observed (Merritt, Chamness, and Allen 1978). Rats were administered ethanol to induce hypothermia and exposed to intense, local MW. BBB permeability was inversely related to ethanol concentration, at a constant level of MW irradiation (Neilly and Lin 1986). Williams and coworkers (1984) found that the BBB was not compromised when whole-body hyperthermia was produced in conscious, unrestrained rats by exposure at either 2450 MHz or ambient heat of 42 ± 2°C. They report that "suppression of BBB permeability occurs, and that this effect is mediated by temperature-dependent changes in endothelial cell function, and not by qualities unique to microwave energy."

This brief review illustrates that microwave-induced changes in the permeability of the BBB have not been consistently observed. Early studies indicated an increase in barrier permeability (Frey, Feld, and Frey 1975; Oscar and Hawkins 1977; Albert 1977; Albert and Kerns 1981). These studies were not replicated by others (Preston, Vavasour, and Assenheim 1979; Gruenau et al. 1982), which was generally attributed to methodologic differences (Justesen 1980; Segal and Magin 1982, 1983; Frey 1983). In an analysis of the literature, Williams et al. (1984) suggest that methodologic limitations in some studies are due to the lack of methods to evaluate effects both physiologically and morphologically. Furthermore, the design of some studies included the use of anesthetics, which may have an impact on the BBB. Later studies found both enhanced permeability at high exposure levels and suppressed permeability. At present, it appears that change in BBB permeability is a nonspecific manifestation of thermal stress.

3.3.4.5 *Interaction with Psychoactive Drugs*

Although the potential for combined interactions with psychoactive drugs was documented in the early 1960s, most of the research in this area has been performed since 1980. A list of compounds derived from selected references is in Table 3-6 (Servantie et al. 1974; Lai et al. 1987; Frey and Wesler 1990).

Michaelson et al. (1961) reported on experiments with dogs exposed at 2.8 GHz and 165 mW/cm^2, finding that the "Thermal response to microwaves is aggravated while under the influence of chlorpromazine, morphine sulfate, or pentobarbital sodium." Baranski and Edelwejn (1968) described interaction of 3-GHz, pulsed MW with various drugs administered to rabbits, although the use of metallic screw EEG connectors could have affected the outcome (Elder and Cahill 1984). A single exposure at 20 mW/cm^2 in animals previously administered chlorpromazine did not prevent EEG desynchronization. Longer exposures (3 h/d, total of 70 to 80 hours) at 7 mW/cm^2 enhanced the effect of CNS-stimulating drugs. Servantie and colleagues (1974) observed that rats, exposed at 4 to 6 mW/cm^2 to pulsed 3-GHz radiation, were less susceptible to the paralyzing effects of curarelike drugs. SARs have been estimated to be around 1 W/kg (Elder and Cahill 1984).

Wangemann and Cleary (1976) exposed unrestrained rabbits and found that administration of sodium pentobarbital prior to irradiation reduced sleeping times. There were no significant differences when MW exposure preceded administration of the stimulant. Lai et al. (1984a) reduced the core body temperature by pentobarbital treatment, then exposed the rats with their heads pointing toward (an-

Table 3-6. Psychoactive Drugs and Compounds

Barbiturates
Hexobarbital
Phenobarbital
Pentobarbital
Drugs
Chlorpromazine
Chloral hydrate
Bemegride
d-amphetamine
Amorphine
Ethanol
Morphine
Naloxone
Chlordiazepoxide
Haloperidol
Curare-like drugs
Cholinergic drugs
Scopolamine
Physostigmine